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GLUTAMIC ACID AND INTELLECTUAL PERFORMANCE

By RUTH WOODS

A relation between nervous and mental functioning and the vitamin B complex has been demonstrated recently and explained on the basis of enzyme systems involved in nerve metabolism.* The unexpected finding that glutamic acid, a commonly occurring amino acid, is capable of raising the intelligence quotient and stimulating emotional and social response in certain limited groups of individuals, is another contribution to present knowledge concerning the biochemical aspects of mental behavior. No explanation is forthcoming as yet for the action of glutamic acid in this respect, but its influence on mental activity remains unmistakable. Whether the activity of glutamic acid is purely a pharmacological one or whether it can be related to dietary factors, such as its possible interraction with other amino acids or other food constituents, remains an unsolved problem of nutrition research.

Glutamic acid is by no means a cure-all for all types of defective mentalities. Even in those cases which respond to treatment, the improvement is limited in degree and the mental gains are soon lost if treatment is stopped. The great significance of this piece of research is the basic finding that mental deficiency is not hopelessly unalterable; that there is a biochemical as well as a psychiatric approach to the treatment of these unfortunate cases. The studies with glutamic acid represent a starting point for this new approach to the understanding of biological mechanisms in relation to mental activity.

Dietary Treatment of Epilepsy:

The present use of glutamic acid had its origin in early studies on epilepsy in children and its control by dietary means. In 1920, an osteopathic practitioner, Hugh Conklin, reported the interesting fact that convulsive seizures in some of his patients were lessened during periods of starvation (1). The following year, this report was confirmed by Geyelin (2) who repeated the experiment with patients at the Presbyterian Hospital in New York. The period of starvation lasted from one to four weeks, water being the only thing taken. Because of the difficulty and impracticality of maintaining patients on such a regimen, a substitute for this method was sought.

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*cf. *Borden's Review of Nutrition Research,* December, 1946 and January, 1947, "Vitamin Deficiency in Nervous and Mental Disease."

While no explanation for the effect of starvation was apparent, it was noted by Geyelin that the beneficial effects of fasting seemed to parallel the formation of ketone acids in the body.

> The ketone acids—acetoacetic acid and β-hydroxybutyric acid, together with acetone, are known as "the ketone bodies." They are formed during the oxidation of fat and, to a lesser extent, of protein. Normally they do not accumulate in the blood stream because they are further oxidized to carbon dioxide and water. Under certain abnormal conditions, however, the ketone bodies are not oxidized as rapidly as they are formed, giving rise to the condition known as ketosis. Such a condition occurs as a result of increased fat metabolism. This may be due to (1) *starvation* during which body fat stores are oxidized; (2) *dietary imbalance* with a low carbohydrate intake so that extra fat (dietary or storage fat) is burned for energy purposes; (3) *diabetes* in which carbohydrate is not utilized, again leading to compensatory use of extra fat for energy. Because both acetoacetic acid and β-hydroxybutyric acid are acids, ketosis is also accompanied by acidosis as well.

Based on the observations in fasting, Wilder (3) devised a special "ketogenic diet" designed to produce a similar accumulation of ketone bodies. The diet was rich in fat and poor in protein* and carbohydrate. It consisted of foods such as bacon, butter, heavy cream, olive oil, egg yolk, etc. Sugar was replaced by saccharin; meat and other proteins were severely restricted. This diet has been used since 1921 with fair success in thousands of cases of epilepsy in both children and adults (4, 5, 6).†

While unquestionably superior to long periods of starvation, the ketogenic diet is a difficult and unpleasant one to follow. Intensive education of the patient is necessary and for complete, reliable control of the diet prolonged hospitalization is essential.

The mechanism involved in the ketogenic diet is not well understood. Several theories have been advanced concerning the importance of ketosis or acidosis, or both, as factors underlying the action of the ketogenic diet (7). In pursuit of the acidosis theory, a search was made for acid-producing drugs as a substitute for the unpleasant ketogenic diet. This led to the discovery, among others, of the marked anticonvulsant properties of the amino acid, *dl*-glutamic acid.

The use of *dl*-glutamic acid was suggested by Waelsch (8) on the basis of the following considerations:

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*A source of body carbohydrate, *cf. Borden's Review of Nutrition Research,* May, 1948, "Modern Concepts of Food Utilization."

†It should be pointed out that the ketogenic diet is only one phase of modern therapy in epilepsy. Other major phases involve the use of sedatives such as phenobarbital and anticonvulsant drugs, notably dilantin. While a discussion of these drugs is beyond the scope of this review, it may be pointed out that their chief value lies in the treatment of the so-called grand mal type of epilepsy. They are less effective in the control of petit mal and psychomotor attacks. It is in the treatment of these latter forms of epilepsy that fasting and the ketogenic diet (and also glutamic acid) are of primary significance.

"It is known that some racemic amino acids, among them *dl*-glutamic acid, give rise to the excretion of the unnatural isomer in the urine when administered to animals. If *d*-glutamic acid (the unnatural isomer*) has a similar inertia in human metabolism, 1 mole of *d*-glutamic acid hydrochloride should furnish 1 mole of hydrochloric acid and two carboxyl groups of which one would be available for acidification. The *l*-glutamic acid hydrochloride also furnishes one equivalent of hydrochloric acid.

"Furthermore, *l*-glutamic acid has been stated to be intimately related to brain metabolism. It is the only amino acid known to be metabolized by slices of brain tissue (9). Brain tissue contains an enzyme which synthesizes glutamine from *l*-glutamic acid and ammonia, decreasing by this mechanism the concentration of free acid in the tissue (10)."

The Effects of Glutamic Acid:

On Convulsions: Accordingly, the use of glutamic acid was attempted clinically by Price, Waelsch and Putnam (8) of Columbia University's College of Physicians and Surgeons. They administered orally an amount of the amino acid, 4 grams, three times a day, sufficient to maintain an acid reaction (about pH 5.0) of the urine. Although anticonvulsant drugs had proved ineffective in the type of seizures involved, these were continued along with the administration of glutamic acid. They found a striking response to glutamic acid in cases of petit mal and psychomotor seizures. No response was obtained in cases of grand mal.

On Social and Intellectual Behavior: Concomitant with the dramatic reduction in number of seizures, an unexpected stimulation of mental and physical response was noted in almost all of the cases described. Psychologic improvement and increased social adjustment were so marked as to excite comment from the patients' families and associates. Withdrawal of glutamic acid was followed by marked regression of mental and physical alertness as well as renewed frequency of seizures. The degree of improvement in mental efficiency, however, was not dependent upon the number of seizures, but constituted an apparently independent response to the amino acid.

The following excerpts from several case histories illustrate the intellectual responses obtained upon administration of glutamic acid:

Case 1. K. M., aged 12 years . . . In addition to the reduction in seizures the patient's parents and instructors in school noted a decided improvement in the patient's personality. He accepted disciplinary measures without prolonged arguments. He was more congenial, cooperative and affable. Peculiar egocentric aptitudes were decreased. Concentration and application in his school work were improved.

*Ed. note.

Case 5. J. R., a youth aged 21, had recurrent psychomotor seizures which were decreased by a combination of drugs. Seizures were prevented by glutamic acid.

Equally encouraging was the subjective improvement. Parents, friends and business associates volunteered the information that the patient's behavior had changed for the better. He was decidedly more affable. His gymnasium instructor reported an improved physical alertness. His assistant noted an increased mental acuity. For the first time he desired and sought social contacts outside the family circle. Hospital attendants noted a decided improvement in cooperation in respect to both hospital routine and clinical tests.

In this case, decided psychologic improvement followed an increased mental and physical alertness, which has aided measurably in social and economic rehabilitation. Seizures recurred at one to four week intervals following withdrawal of the drug irrespective of supplementary medication after the onset of the aura. Though the major portion of the psychologic improvement has been maintained, there has been no progress on other forms of medication.

Case 7. M. B. S., a woman aged 41, had onset of seizures at the age of 20. Severe social, educational and economic restrictions have been a serious handicap. Seizures were strikingly reduced by phenytoin sodium. Personality disturbances combined with social maladjustment prevented a satisfactory rehabilitation following control of the seizures. Severe emotional instability is far more incapacitating than infrequent psychic equivalents, grand mal, petit mal and psychomotor attacks.

Improved mental and physical alertness was noted objectively and subjectively. Tolerance of the opinions of others was noticeably improved. Insight was acquired into her pronounced introversion, egocentric responses to minor stimuli, recurrent anxiety states and mild depressions. The efficiency with which she performed the details of her position as a librarian was augmented by the increased mental alertness. Gymnasium exercises became less laborious.

In a subsequent study, Waelsch and Price (11) confirmed their findings with respect to personality improvement in epileptic persons treated with glutamic acid. They were able further to narrow down the effect of the racemic *(dl)* mixture to the naturally occurring *l*-glutamic acid which is an important constituent of both food and body proteins.

Animal Experiments in Learning with Glutamic Acid:

The observations on increased physical and mental alertness in epileptic patients following the administration of glutamic acid raised the question as to the possible role of glutamic acid in mental processes not associated with convulsive seizures. This led to several animal experiments designed to determine the influences, if any, of glutamic acid on the learning process in rats.

In one of these studies, reported by Zimmerman and Ross (12), rats were fed glutamic acid or derivatives as supplements to a normal adequate diet. The ability of these animals to find their way through a maze was

compared with that of a control group receiving an adequate basal diet without glutamic acid. Performance was measured by the number of trials necessary to complete the maze, number of errors made (i.e. running into cul-de-sacs) and by the time necessary to complete the runs. The average number of trials necessary to reach perfection was 3.9 in the rats receiving glutamic acid and 12.1 in the controls; the number of errors, 8.5 and 36.5, respectively; and the total time approximately 290 and 666 seconds, respectively. Animals fed the amino acid, aminoacetic acid, showed no improvement over the control group, thereby indicating that a specific action of glutamic acid, and not the effect of an increased supply of nitrogenous compounds, is involved.

Six months later, results of another animal study were published by Albert and Warden (13) of the Department of Psychology at Columbia University. Their experiment was undertaken to determine whether the addition of *l*-glutamic acid to normal diets would not only improve speed of learning, but permit rats to learn more complex problems than rats fed only a normal diet. In other words, could glutamic acid supplements permit rats to attain a higher intelligence level than ordinarily possessed by that species?

The test was conducted by using a specially devised problem box (14). This consists of a circular cage with a series of floor plates to be stepped upon by the animal in a given sequence in order to secure food. The animal passes gradually from a simple level (one plate) to increasingly difficult ones (two to ten or more plates). When the pattern becomes too difficult the animal fails and the last step learned is taken as the level score of the individual. The following table summarizes the results:

	Glutamic Acid Group		*Control Group*	
Setting	Total number	Number successful	Total number	Number successful
1-Plate	9	9	8	8
2-Plate	9	9	8	4
3-Plate	9	8	8	2
4-Plate	9	1	8	0
5-Plate	9	1	8	0
6-Plate	9	0	8	0

As can be seen, the glutamic acid group made better showings in several respects. All of the glutamic acid group succeeded at the 2-plate level, whereas only half of the controls were able to master the problem. Again, all but one of the test group passed the 3-plate problem while only one-quarter of the controls succeeded. None of the controls went beyond the 3-plate stage, whereas one of the glutamic acid group was able to master

both 4 and 5-plate stages. Although the number of animals used was small, the performance of the controls is in general agreement with that reported elsewhere (15) for a larger group of normal animals. In this latter report, none of 33 rats was able to go beyond the 3-plate level in the same apparatus.

Glutamic Acid in Mentally Retarded Human Subjects:

The improvement in mental efficiency noted in epileptic patients—a phenomenon definitely not related in degree to the therapeutic success of glutamic acid in decreasing convulsions—plus the encouraging results of learning experiments in animals led to the thought that this amino acid might prove similarly effective in human cases of mental deficiency not associated with epilepsy. There was little concrete basis for such a hope because (a) intellectual functions in rats and humans cannot be compared and (b) the patients with epilepsy were all of normal intelligence and the effect of glutamic acid seemed to be more conspicuous in influencing social behavior than in changing the intelligence level. Nevertheless, because of the unmistakable implication of glutamic acid in brain metabolism, an investigation into the possibilities of improving intelligence levels was made.

The first of these was reported in 1946 by Albert, Hock and Waelsch (17). They gave glutamic acid to eight persons suffering from mental deficiency. The chronological ages ranged from 6 to 26 years, the mental ages from 2 to 8 years. The I.Q.'s ranged from 22 to 73. The subjects were given a battery of intelligence tests adapted to their individual requirements. Tests were given under optimum conditions and after disposal of all possible interfering factors. After the I.Q. level of the patients was established they were given glutamic acid or placebos and retested at intervals. The rise or fall in I.Q. was compared with the original test level.* A significant rise of mental age was found during administration of the amino acid. The gains in I.Q. disappeared when the patients were transferred, unknown to themselves to placebos. Restoration of glutamic acid therapy again increased the I.Q.

> In one case, for example, a 14 year old boy able to speak only two or three word phrases was found to have an I.Q. of 42. In seven and a half years prior to glutamic acid therapy the boy's mental age had increased 2 years and 5 months. During glutamic acid treatment for only two months, the patient gained 1 year, 5 months in mental age or two-thirds of his entire gain during the previous seven and a half years. The I.Q. then dropped to 43 when placebos were substituted for glutamic acid.

*The accuracy of the original test level was confirmed by record of previous years from which no deviation, or that of one point only, had occurred in repeated I.Q. tests.

Another patient, aged 18, was found to have a mental age of 7 years, 10 months. After one month on glutamic acid, his mental age increased to 9 years; after 2½ months, to 9 years, 8 months. Eventual return to lower I.Q. levels followed discontinuation of glutamic acid administration.

As evidence against the argument that the gain in I.Q. was not valid but resulted from an increased familiarity with the tests, the authors point out:

(1) The patients were tested several times prior to glutamic acid administration and none of them showed a significant variation in I.Q. These pre-treatment tests were spaced at the same time intervals as those carried out later during the glutamic acid periods.

(2) Each time the patient was retested, not only the original Binet but other tests with which the patient was not familiar were used.

(3) The best proof, however, rests in the fact that the patients dropped in I.Q. during the placebos period and regained the loss again when glutamic acid was administered.

Following these encouraging studies, a similar report appeared one month later by Zimmerman *et al* (18). Seven of the nine subjects in this study were patients with convulsive disorders; two were mentally retarded without convulsions. A series of standardized intelligence tests was given at the beginning and at the end of a six month period during which the patients received *l*-glutamic acid in doses of about 12 grams per day. As in the previous study, sharp improvements in the mental age were noted, the children progressing at a rate faster than would be expected of normal children. Control subjects were given anticonvulsants other than glutamic acid. From the combined responses of both groups, it was felt that part of the improvement was due to reduction of seizures.

Both of these preliminary studies demonstrated improvement in personality as well as in verbal ability and performance. Dull and inattentive patients, after receiving glutamic acid, became more alert, attentive and persistent in trying to solve the test problems given them.

The results have been interpreted generally as being due to a better use of existing facilities rather than an acquisition of new intellectual tools (increases in intelligence). In other words, it is believed that glutamic acid, a substance affecting brain metabolism, probably aids patients whose intellectual potentialities were basically normal, but have been damaged by brain disease or injury, or inhibited by emotional mechanisms.*

*Glutamic acid has also been found to be successful in overcoming the loss of memory and emotional depression resulting from electroshock treatment of psychiatric disorders.

Mechanism of Glutamic Acid Activity:

In their further studies on the effectiveness of glutamic acid in epileptic condition, Waelsch and Price (11) attempted to explore the possible biochemical mechanisms underlying the behavior of this amino acid. The original theory behind the use of glutamic acid had been based on two points: (1) The close relationship of *l*-glutamic acid to brain metabolism and (2) the acidifying tendency of the racemic mixture, *dl*-glutamic acid.

Studies were made on the influence of the administration of *dl*-glutamic acid hydrochloride on the acid-base balance of the blood and urine and on its metabolic fate in the human subject. No shift in the acid-base balance of the blood following the administration of *dl*-glutamic acid hydrochloride could be detected with the methods applied and the decrease in seizures occurred too soon for it to appear likely that this was the effect of a mildly acidifying agent. The results of these studies, combined with observations on the effect of administration of the *l*-glutamic acid isomer, indicated strongly that the therapeutic value of the amino acid might be ascribed in large part to *l*-glutamic acid alone* and not to the acidifying properties of the racemic mixture. The question still remaining is—to what property of *l*-glutamic acid can its behavior be attributed?

At present there is no answer to this question. There are, however, some clues as to the direction which future research may take in the quest for the solution to this problem. In the experiments of Zimmerman and Ross (12) and Albert and Warden (13) the effect of glutamic acid or its derivative (*dl*-pyrrolidincarboxylic acid) on improving learning processes definitely implicates glutamic acid in some way, direct or indirect, with cortical activity† of the brain.

In a recent investigation of the biochemical activity of glutamic acid, Nachmansohn, John and Waelsch (16) showed that glutamic acid has an adjuvant action on the enzyme system which synthesizes acetylcholine, a compound intimately involved in nerve metabolism and the transmission of nervous impulses. After the acetylcholine-synthesizing enzyme extract was dialyzed, a decreased ability to manufacture acetylcholine was found. The synthesizing power was found to be partially restored by the addition of glutamic acid (or of citric acid) to the extract.

As pointed out by Waelsch and Price (11), these observations which point to the specific involvement of glutamic acid in biochemical mechanisms intimately connected with the function of nervous tissue, may become

*Without the unnatural glutamic acid isomer and without any other anticonvulsant drugs.
†The cortex is generally believed to be the seat of learning in the brain.

of increased importance if there should be found a deficiency of glutamic acid or substances metabolically related to it in patients with petit mal or certain types of mental deficiency.

Dietary Significance of Glutamic Acid:

The use of glutamic acid in these experiments has been of a clinical nature involving the separate administration of *l*-glutamic acid or of *dl*-glutamic acid hydrochloride. It is particularly significant that glutamic acid is a naturally occurring amino acid. It is a major component of food and body proteins. For example, casein contains 24% glutamic acid; beef protein, 15%; wheat gliadin, 42%; corn zein, 35%; soy bean meal, 19%. It is interesting to note further that mono-sodium glutamate, a salt of glutamic acid, is widely used as a flavoring agent. This material was originally used in the Orient to add a meat-like flavor to vegetable foods. Its introduction in the United States was in the form of "soy sauce." At the present time, its use has increased to millions of pounds annually in the form of various flavoring sauces and in the industrial processing and canning of such common foods as soups, etc.

A daily protein intake of 70 grams would correspond roughly to an intake of glutamic acid of 7 to 10 grams. The therapeutic dose of 5 grams per day represents an additional 50% of the dietary intake. This amount could be provided by the addition to a normal diet of approximately a quart of milk or 8 slices of bread. The answer to the question as to whether an extra quart of milk or 8 extra slices of bread will substitute for glutamic acid capsules lies in future experiments. It is possible of course that such dietary supplementation might fail due to an increase in carbohydrate intake. Furthermore, the importance of direct glutamic acid therapy may lie in changing the ratio of glutamic acid to other compounds. The use of food as a source of glutamic acid might raise the absolute intake of glutamic acid without changing the ratio.

Conclusion:

Clinical and experimental evidence, in large measure, has contributed favorably to the recognition of the role of the amino acid, glutamic acid, in improving social behavior and intellectual performance in certain types of secondary mental deficiencies. These include depressed intellectual levels associated with epilepsy as well as certain types of mental deficiency, such as feeblemindedness or imbecility. The mechanism of the action of glutamic acid is unknown, although this amino acid is known to be involved in brain

metabolism. The studies reported constitute only a fraction of the cases which have been, and still are, undergoing treatment with this amino acid.

As has been concluded by Albert *et al* (17):

> "We do not want to raise any extravagant hope that cases of secondary mental deficiency and, by implication, other forms of feeblemindedness, may be cured with glutamic acid; but there is some indication that a limited influence can be demonstrated on some of these patients. Mental defects have been considered unalterable, and research and treatment have been concentrated on the social and educational aspects, which are of great importance. Nevertheless, we felt that attempts should be made to test the possibility of improving brain function by biochemical means. The results obtained with glutamic acid are considered to be a starting point for further investigations. Long and tedious research will be necessary to solve the question of the specificity of glutamic acid and to test other substances which may benefit some patients and may also elucidate basic neural mechanisms in relation to mental activity."

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NEWS DIGEST

VITAMIN C IN CABBAGE — In Canada the consumption of cabbage ranks next to that of potatoes and carrots. The vegetable is an excellent source of ascorbic acid, the average value being about 59.2 mg. per 100 grams, as compared with 52.4 mg. per 100 grams for fresh oranges. It has been pointed out that for low-income groups who may be unable to purchase citrus fruits or juices, cabbage, *especially if eaten raw,* can serve as a major source of vitamin C in the diet. Recent studies on the effect of cooking on the vitamin C content, however, reveal large losses of the vitamin. In large quantity cooking, either by boiling or steaming, about 70 per cent of the vitamin C is lost; in small quantity cooking, the loss is about 60 per cent. Holding of cooked cabbage on steam tables in restaurants results in a loss of over 90 per cent of the vitamin C content after two hours. As a result it is suggested that cabbage be eaten raw, in the form of salads, in order to obtain maximum vitamin C value; that cooked cabbage in restaurants be prepared on a staggered system, i.e. in small quantities suitable for immediate serving, to eliminate loss during holding on a steam table. An additional finding of interest was the fact that the highest concentration of ascorbic acid is not only in the green outer leaves (usually discarded), but to an even greater extent in the core which is also usually discarded. — *J. Am. Diet. Assoc. 24,* 101 (1948).

MPF — MPF, brief for Multi-Purpose Food, is a newly developed concentrated emergency food. It consists of soya grits, onion powder, hydrolyzed yeast, salt, herbs, spices, vitamins and minerals. One 2-oz. portion costs only 3 cents (as of Nov. 1947) and is said to be equivalent in nutritive value to a full meal consisting of ¼ lb. meat, a glass of milk, and one normal serving each of peas and potatoes. The nutritive composition is:

Protein	42.3	per cent
Fat	4.51	per cent
Carbohydrate	35.05	per cent
Calcium	0.46	per cent
Iron	0.0123	per cent

Because MPF is low in caloric value and lacking in ascorbic acid, it is suggested that when used to make up a meal, bread and an ascorbic acid supplement be added. MPF, a dried canned product, a palatable product itself, takes on the flavor of any food cooked with it and is thus valuable as an extender.—*N. Y. Times, November 30,* (1947).

VITAMIN A DEFICIENCY IN BEEF CATTLE: Prolonged vitamin A deficiency in cattle may result in generalized edema. The condition is often referred to as anasarca. This report presents statistics showing that 651 beef carcasses were condemned for anasarca by federal meat inspectors during the period of July, 1941, to December, 1946, inclusive. Other symptoms of vitamin A deficiency were found to be prevalent among cattle with anasarca in the corn belt area. Occurrence of symptoms resulted after a long fattening period in dry lot, when the cattle were fed either stored or new corn in combination with a roughage of low carotene content, such as oat hay or straw. Cases of anasarca were readily produced experimentally by feeding a carotene-deficient ration or a grain ration containing 80 per cent of a new-crop yellow corn together with oat straw as roughage. By providing a source of carotene in the feed the disease was alleviated.—*J. Nutr. 34,* 603 (1947).